

In our review of the literature, we found many drugs described in association with EM (antibiotics, corticosteroids, nonsteroidal anti-inflammatory drugs), but there were few reports linking EM to topical treatments.¹⁻³ We detected just 3 cases of EM associated with topical imiquimod, and none of the patients had Gorlin syndrome. Systemic absorption could explain why topical imiquimod causes EM, as the immunomodulatory effects of the drug could trigger a type III and/or IV hypersensitivity reaction, ultimately leading to EM. An intense local inflammatory reaction such as that experienced by our patient would probably favor this systemic absorption, predisposing patients to an EM-type skin eruption. Nonetheless, whether or not patients with Gorlin syndrome have an immune-based predisposition to EM remains to be confirmed.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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Treatment of Livedoid Vasculopathy With Rivaroxaban: A Potential Use of New Oral Anticoagulants for Dermatologists[☆]



Vasculopatía livedoide tratada con rivaroxabán. Potenciales usos de los nuevos anticoagulantes orales para el dermatólogo

To the Editor:

Livedoid vasculopathy (LV) is a noninflammatory thrombotic disease that affects the small blood vessels of the skin and

is characterized by livedo racemosa and painful skin ulcers on the lower extremities.^{1,2} We report 2 cases of LV in which treatment with rivaroxaban achieved a full and sustained response. We also review novel oral anticoagulants with potential applications in dermatology.

Case Description 1

A 53-year-old woman with no relevant past history presented with multiple skin ulcers on her feet. The ulcers were painful and had been present for 2 years. Physical examination revealed an ulcer measuring approximately 3 cm on the medial aspect of the left foot against a background of livedo racemosa and retiform purpura (Fig. 1A). The patient's



Figure 1 A, Cutaneous ulcer on the medial aspect of the left foot against a background of livedo racemosa and retiform purpura. B, Atrophic blanche due to scarring following the use of oral rivaroxaban.

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medical history and exploratory tests ruled out hypercoagulability, systemic inflammatory disease, and infection. In the evaluation of peripheral arterial disease, no alterations were observed in the ankle-brachial index or in the Doppler ultrasound scan. Skin biopsy showed fibrin clots in the dermal vessels, extravasated red blood cells, hyalinization of the vessel walls, and neovascularization consistent with LV. Treatment was initiated with acetylsalicylic acid 300 mg/d, intravenous alprostadil 60 μ g/d, and subcutaneous enoxaparin 1 mg/kg/d. These treatments resulted in a mild reduction of pain and had almost no effect on ulcer healing. The patient was subsequently started on rivaroxaban 10 mg/d, which resulted in full resolution of pain after a week and healing of the ulcer at 3 months (Fig. 1B). Twelve months after initiation of treatment with rivaroxaban, the patient was free of pain and ulcers, although there were no changes to the livedo racemosa.

Case Description 2

A 55-year-old woman with no past history of interest presented with multiple painful cutaneous ulcers of 12 years' duration on the lower legs. Physical examination revealed multiple ulcers, some measuring up to 10 cm, with a necrotic base and a livedo racemosa pattern extending up to the knees (Fig. 2A). Based on the patient's medical history and tests, a secondary cause was ruled out. Skin biopsy findings were consistent with LV. The lesions were refractory to treatment with oral prednisone, hydroxychloroquine, acetylsalicylic acid, and pentoxifylline. Treatment with rivaroxaban 10 mg/d led to reversal of pain after 2 weeks and full resolution of ulcers by month 4 (Fig. 2B-D). After 9 months of treatment with rivaroxaban, the patient remained pain free and ulcer free. Similarly to the first case described, there were no changes to the livedo racemosa.



Figure 2 A, Cutaneous ulcers against a necrotic background on the legs. B-D, Progressive healing of ulcers after initiation of oral rivaroxaban.

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